Cardiac output monitoring

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Abstract

Cardiac output monitoring has become commonplace amongst the standard monitoring in anaesthesia for major surgery and in intensive care. Multiple techniques are used, employing different models to calculate flow, which cannot be directly measured. Each method has differing advantage and disadvantage profiles and so the choice of device should consider a balance of invasiveness, usability, reliability and validity. Trend monitoring is generally emphasized over absolute values.

Keywords Aortic Doppler; bioimpedance; cardiac output; echocardiography; indicator dilution; oesophageal Doppler; pulse contour analysis; pulse waveform analysis; thermodilution

Royal College of Anaesthetists CPD Skills Framework: Scientific principles.

Learning objectives

After reading this article, you should be able to:

- understand the importance of cardiac output monitoring in anaesthesia and intensive care
- understand the physiology and physics behind the commonly used methods of cardiac output monitoring – Fick principle, pulse contour analysis, bioimpedance, aortic Doppler and echocardiography
- understand the strengths, common uses and limitations of the above methods of measuring cardiac output

Introduction

Cardiac output (CO) is an important parameter, representing the total cardiovascular blood flow through the heart. It is defined as the volume of blood ejected from the right or left ventricle, per unit time commonly being described as litres per minute.

CO = Stroke volume (SV) × Heart rate (HR)

Oxygen delivery is vital to tissues, especially in critical illness or post-surgery where oxygen demand is increased due to the inflammatory response. To ensure adequate organ function and aerobic respiration in these situations perfusion and oxygen delivery must be maintained. Delivery of oxygen is a result of both the cardiac output and arterial oxygen content:

DO_2 [ml/minute] = **Cardiac output** [litres/minute] × **Arterial oxygen content** [ml/dl] × **10**

Therefore, by utilizing techniques to calculate the CO the information can be combined with the measurable arterial oxygen content to give a good estimation of oxygen delivery to tissues, impacting how the patient may be treated.

For almost a century there have been a number of ways to calculate cardiac output, with each in turn having issues around potential sources of error, cost or limitations of use. Most forms of CO measure are limited to controlled areas such as critical care or the operating theatre due to their invasive nature. Each technique can be potentially classified into three main types:

- Invasive: utilizes a central line, arterial line or pulmonary catheter.
- Minimally invasive: ultrasound techniques exploiting the oesophagus' proximity to the aorta.
- Non-invasive: a number of techniques are used including bioimpedance.

All these methods aim to accurately infer the stroke volume of the patient to give a value for the CO.

This article aims to explain the principles and physiology behind each of these methods and include the limitations of each.

Invasive methods of analysis

Fick principle

This principle holds that the uptake or excretion of a substance by an organ is equal to the difference between the amount of substance entering the organ and the amount leaving (Figure 1).

$$\text{Blood flow to an organ} = \frac{\textbf{O}_2\textbf{ consumption (i.e. rate of addition or removal of substance)}}{\textbf{Concentration difference (arterial} - \textbf{venous)}}$$

It is important to realize that because the entire blood volume passes through the lungs, changes in oxygen concentration can give an accurate picture of the CO in a patient. However, the use of oxygen is not practical because it requires a pulmonary artery (PA) catheter and also makes assumptions about the system within which it is estimating concentration difference (e.g. that the pulmonary and systemic volumes are equal). Additionally, to properly ensure accurate measurement of oxygen a sealed hood is required for measuring gases, which again adds complexity to utilizing oxygen as the measured substance in the Fick principle.

Subsequently, dye or thermodilution techniques are utilized instead and assume that the rate of decay in the concentration of the substance/temperature change is proportional to the flow rate through the organ.

Dye dilution techniques

Method of use: in this instance a known volume/amount of a marker substance is chosen and injected into the pulmonary artery or central vein (therefore it requires a central venous catheter (CVC) or PA catheter). A peripheral arterial line is then used to continuously sample the concentration of the dye. This then can produce a graph which measures the concentration over time (Figure 2).

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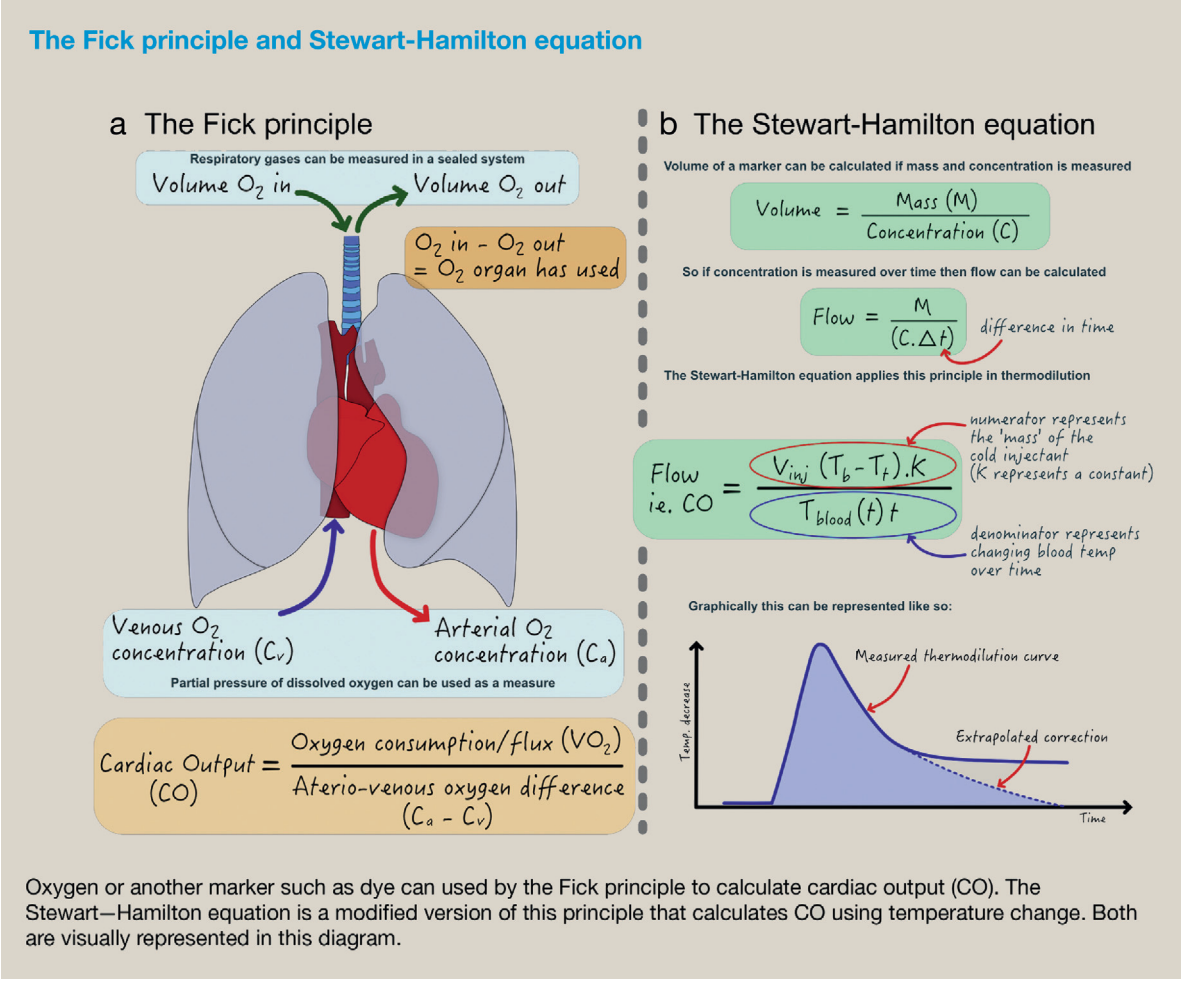
The Fick principle and Stewart-Hamilton equation
a The Fick principle
Respiratory gases can be measured in a sealed system
Volume O_2 in
Volume O_2 out
O_2 in - O_2 out = O_2 organ has used
Venous O_2 concentration (C_v)
Arterial O_2 concentration (C_a)
Partial pressure of dissolved oxygen can be used as a measure
Cardiac Output (CO) = Oxygen consumption/flux (VO_2) / Aterio-venous oxygen difference (C_a - C_v)
b The Stewart-Hamilton equation
Volume of a marker can be calculated if mass and concentration is measured
Volume = Mass (M) / Concentration (C)
So if concentration is measured over time then flow can be calculated
Flow = M / (C.Δt)
difference in time
The Stewart-Hamilton equation applies this principle in thermodilution
Flow ie. CO = V_{inj} (T_b - T_f).K / T_{blood} (t) t
numerator represents the 'mass' of the cold injectant (K represents a constant)
denominator represents changing blood temp over time
Graphically this can be represented like so:
Measured thermodilution curve
Extrapolated correction
Temp. decrease
Time

Oxygen or another marker such as dye can used by the Fick principle to calculate cardiac output (CO). The Stewart—Hamilton equation is a modified version of this principle that calculates CO using temperature change. Both are visually represented in this diagram.

Figure 1

Traditionally, indocyanine green was the dye used to create this graph as it had a low toxicity and short half-life. This limited the amount of recirculation after the initial passage through the systemic vasculature. Lithium is now more commonly used as it is not an ion present normally in plasma so only very small doses are required to gain a high signal to noise ratio, again this ensures patients are not exposed to toxic or pharmacologically significant doses.

CO can then be calculated from the graph produced by using the area under the curve (integral of the curve) and a modification of the Fick equation known as the Stewart—Hamilton equation (Figure 1).

Limitations

- Logarithmic transformation of the concentration is necessary to give a more accurate estimation of the area under the curve and mitigate for recirculation of dye.

Thermodilution

To avoid the problem with potential recirculation of dye, a thermodilution technique was developed. This then became the 'gold standard' against which other CO measures are compared to. It utilizes the same principle as dye dilution except it uses 10—15 ml cold saline which is then injected though a PA catheter. The change in blood temperature is then measured from the proximal port where it's injected to the thermistor in the distal portion of the catheter. A modification of the Stewart—Hamilton equation is used to calculate the CO (Figures 1 and 2).

Method of use: it is inserted by inflating a balloon at the tip of a PA catheter and 'floating' the catheter through the right atrium, the right ventricle and letting it rest in the pulmonary artery (shown in Figure 2). The cold saline is injected and CO calculated.

Benefits

- It requires no blood sampling and there is no recirculation issue as the blood has warmed back up to normal by the time it reaches the pulmonary circulation.
- This system continues to remain accurate in the presence of balloon pumps and arrhythmias and any inaccuracies can be reduced by taking multiple measurements.

Complications

- Those linked to pulmonary catheter insertion including arrhythmias, infection, valvular damage and pulmonary artery rupture.

A study (PAC-MAN study group) in 2005 aimed to assess the mortality associated with PA catheter insertion in critically ill

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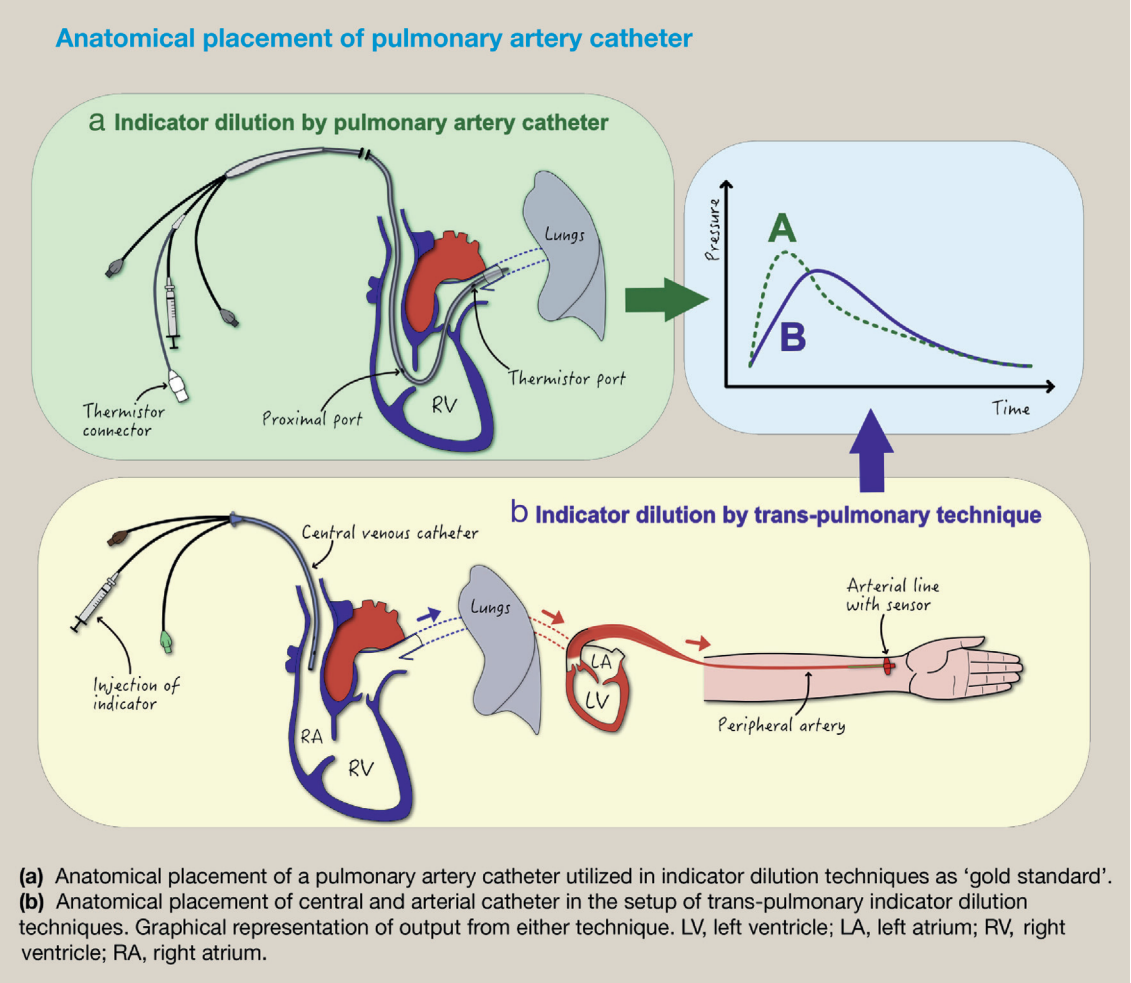

(a) Anatomical placement of a pulmonary artery catheter utilized in indicator dilution techniques as 'gold standard'. **(b)** Anatomical placement of central and arterial catheter in the setup of trans-pulmonary indicator dilution techniques. Graphical representation of output from either technique. LV, left ventricle; LA, left atrium; RV, right ventricle; RA, right atrium.

Figure 2

patients.[1] Despite this randomized controlled trial finding no evidence of benefit or harm, there has not only been a move to less accurate methods of measuring CO, but indeed a move towards their cessation of use in clinical practice. The reasons for this change are multifactorial and extend beyond the remit of this article; however, recognizing this form of CO monitoring as the 'gold standard' by which others are measured is important.

Pressure waveform analysis

The clinical use of pulse pressure, assessing its character and volume, has been utilized long before more quantitative measures in current practice. This has been achieved with the use of arterial cannulation and the assessment of a pulse pressure waveform and now form the most widely used techniques for monitoring cardiac output.

Windkessel effect: the left ventricle (LV) contracts generating a systolic pressure and typically ejects approximately 70 ml (the stroke volume) of blood into the aorta.

Blood within the aorta then causes the walls to expand and stretch to accommodate this new volume increasing the pressure from 80 mmHg to 120 mmHg. The ejected blood has kinetic energy that propels it onwards into the systemic vasculature while the stretched elastic walls of the aorta have potential energy. This is then converted to kinetic energy when the walls recoil and helps ensure that blood continues to be moved though the systemic vasculature in diastole and helps maintain a diastolic BP. The phenomenon described as the Windkessel effect is how this sinusoidal pressure generated in the heart is then converted into a positive and constant pressure at the tissues. Otto Frank, the German physiologist who first described this phenomenon, likened it at the time to the 'Windkessel' air chamber used in fire engines that converted the sinusoidal pump pressure generated into a constant water stream (Figure 3).[2]

This generation of a constant pressure invariably becomes more difficult for the body as it ages with the loss of elastin and collagen fibres in the arterial walls. Loss of compliance and stretch means the systemic circulation struggles to compensate for SV and the major increase in pressure during systole. Subsequently this helps explain why systolic hypertension becomes more prevalent in a more elderly population.

What does the arterial pressure wave show? There is a difference between an arterial pressure wave and a blood flow wave. When feeling a pulse it is the pressure wave that is being felt and the blood flow is assumed. Similarly the graph generated using an arterial cannula shows a pressure wave and the blood flow is inferred (Figure 3).

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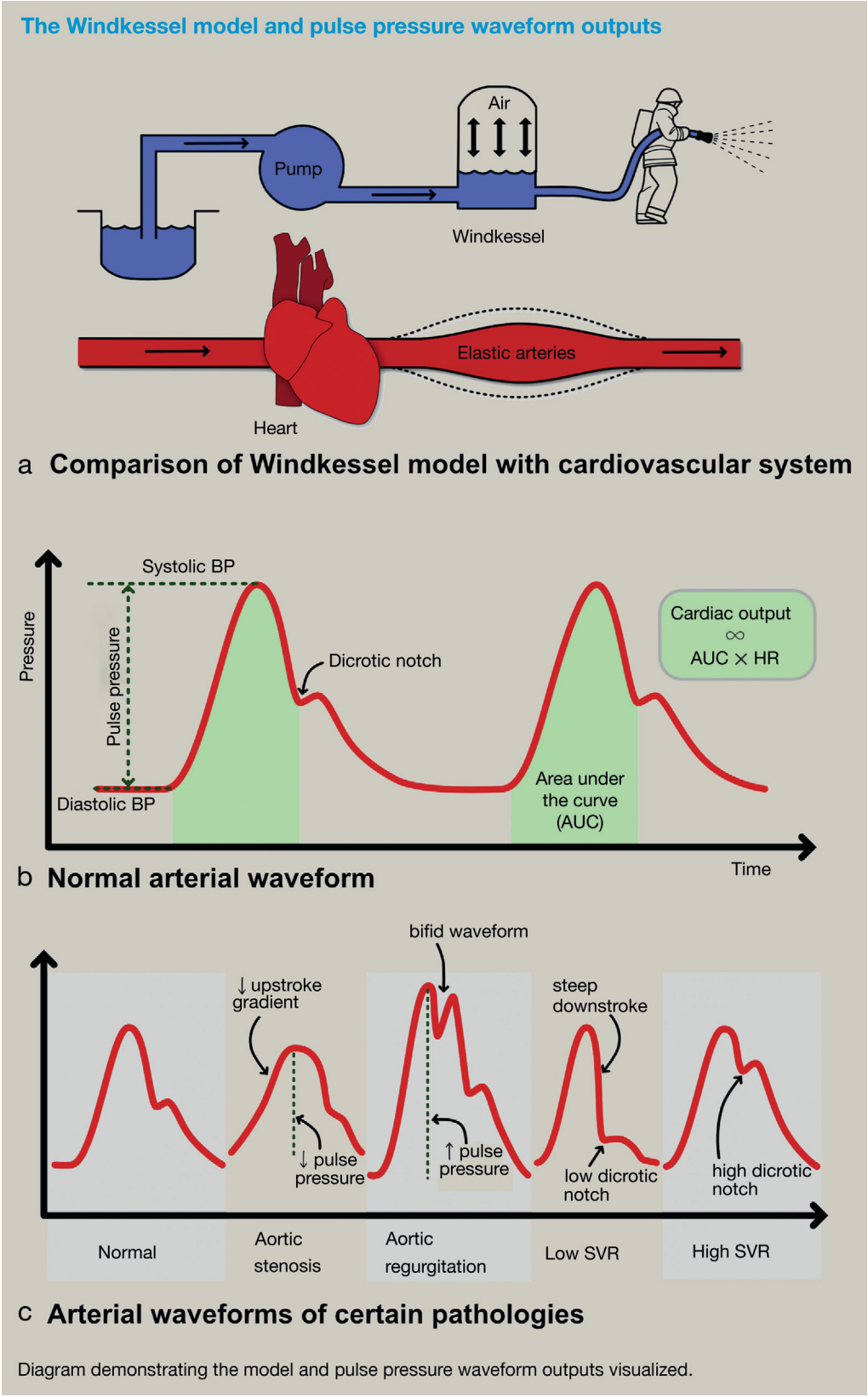

Diagram demonstrating the model and pulse pressure waveform outputs visualized.

Figure 3

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As the LV ejects the stroke volume into the aorta, the intra-aortic pressure rises to a peak of classically 120 mmHg (SBP) and as the ejection stops and elastic recoil occurs the pressure drops to a trough at about 80 mmHg (DBP). The interruption to this curve occurs when the aortic valve closes and there is another short increase in pressure within the aorta — this is the dicrotic notch.

The waveform seen will alter depending on the location of the artery within which it is being measured for example within the aorta itself versus a more peripheral artery.

What information can we get from the pulse pressure waveform? Pulse pressure variation (PPV) is a measure of the difference in the maximum and minimum pulse pressures seen during a respiratory cycle. This can only be utilized in positive pressure ventilated patients as the venous return to the heart is affected by the positive pressure insufflation of the chest in under-filled/hypovolaemic patients altering the preload with each breath. This causes a 'swing' or variation over time in the pulse pressure. Specific monitoring can calculate a percentage PPV or alternatively you can attempt to visualize a variation. More than 10% would suggest a patient is under-filled.

The slope of the waveform gives an indication of cardiac contractility as an increase in gradient or upstroke of the curve suggests a greater pressure generated by the heart over time. This normally can be interpreted as increased contractility, but in the context of aortic valve disease this may not be accurate. Aortic stenosis can reduce the upstroke gradient as there is a fixed outlet into the aorta, while in regurgitation a normal contractility can demonstrate a bifid waveform (Figure 3).

Systemic vascular resistance (SVR) is demonstrated by the downstroke of the waveform. Low SVR is indicated by a steep downstroke with a low dicrotic notch giving the appearance of a thick and pointed waveform. Conversely a dicrotic notch and much less steep downstroke is indicative of a higher SVR (Figure 3).

Pulse pressure contour analysis

The information described above therefore allows the continuous assessment of cardiac output by multiplying the area under the curve by the heart rate. A computer algorithm specific to the system in use then calculates the cardiac output along with a number of other measured or derived variables, which includes the SV, cardiac index (CI) and stroke volume variation (SVV).

SVV is a subtle but different measure to PPV. Again, this measure is affected by venous return in the respiratory cycle as intrathoracic pressure changes. In this instance the actual SV is calculated using the algorithm and the difference between the maximum and minimum SV over time is determined. This gives a measure of fluid responsiveness based on the assumption that if you are 'more full' your venous return varies much less and this translates to the SV. In practice a patient with an SVV >15% is likely to increase their CO with a fluid bolus whereas if it is <10% then this is less likely.

These analysis systems can be divided into two different types—calibrated and uncalibrated. In general, the calibrated systems use the Fick principle described in the last section to give an estimation of CO by calibrating that with the pulse pressure waveform in the algorithm. Uncalibrated methods use population data based on demographics to calculate the likely values in its algorithm.

Calibrated systems

Pulse contour cardiac output (PiCCO)

This system uses a transpulmonary thermodilution method to calibrate its algorithm (Figure 2).

Method of use: it requires a standard central line and a thermistor-tipped arterial line which is placed within the femoral or brachial artery. Cold saline is injected into the central line and the temperature change is measured at the arterial line. This information is then utilized by the program that analyses the pulse pressure waveform to calculate the cardiac output and ensure accuracy of the algorithm.

Limitations

- Inbuilt margin for error (compared with the pulmonary artery catheter thermodilution technique) as heat is lost and dissipates as it passes through the pulmonary circulation.

Lithium dilution cardiac output (LiDCO)

This system uses a lithium dilution method to calibrate its algorithm and requires only a standard arterial line.

Method of use: lithium chloride is injected into a central or even peripheral vein and the change in concentration is measured by an electrode sampling at the arterial line site. This removes the challenge experienced by PiCCO with the loss of heat in the lungs, however it requires recalibration every 8 hours or after a major change in the haemodynamic state of the patient.

Limitations

- Cannot be used by patients who are taking therapeutic lithium as it cannot be calibrated.
- Muscle relaxants can cross-react with the lithium measuring electrode and render it inaccurate.
- Frequent calibrations over time needed, due to repeated doses of lithium, again reducing the accuracy of the system.

Uncalibrated systems

FloTrac/Vigileo

In this system a specialized pressure sensor known as FloTrac is used.

Method of use: the FloTrac sensor is attached to a standard arterial line with this connected to a Vigileo monitor which uses data based on population demographics (e.g. age, weight, height and sex) to estimate aortic compliance. This information is then input into the FloTrac/Vigileo pulse pressure analysis algorithm to calculate CO.

Limitations

- Uncalibrated and relies on population demographic data. Therefore a number of assumptions about anatomical and physiological measurements based on 'average' data which may not be applicable or accurate in certain patients.

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LiDCOrapid

This system uses the same pulse pressure algorithm to calculate CO as the LiDCO system, but importantly utilizes nomograms based on demographic information rather than lithium dilution. Similarly this can lead to inaccuracy and error in CO estimation.

As a whole pulse pressure waveform analysis does show a good correlation with the 'gold standard' PAC thermodilution method of measuring CO.[3] Importantly there are certain conditions and situations where this is not the case and can render pulse pressure analysis inaccurate.

Limitations to pulse pressure contour analysis

- Clinical conditions and situations where pulse pressure analysis is inaccurate include:
  - under- or over-damped arterial line trace
  - cardiac arrhythmias
  - aortic regurgitation
  - the presence of an intra-aortic balloon pump.

Benefits to pulse pressure contour analysis

- Fairly accurate measure that reflects well the accuracy of thermodilution[3] while avoiding the invasive complications of a PA catheter.
- Easily accessible in theatre or critical care environments.
- Utilizes equipment that is already often in place, namely an arterial catheter making it more cost effective.

Complications

- Nil significant other than those associated with arterial cannulation.

Minimal and non-invasive methods of analysis

Aortic Doppler

Principles of ultrasound and the Doppler effect: aortic Doppler is a method of deriving cardiac output based upon the velocity of red blood cells in the aorta as a measure of aortic blood flow. A probe is inserted into the oesophagus and directed towards the descending aorta. It utilizes ultrasound and the Doppler principle, which it would be worth revising here. Ultrasound probes contain piezoelectric crystals that emit sound waves at a set frequency (4–5Hz in the oesophageal Doppler) when electricity is applied to them. These high-frequency sound waves are able to penetrate tissues, which can be directed at an interface of structures of differing density. A proportion are then reflected back and reach the piezoelectric crystals in the probe which convert the kinetic energy back to an electrical signal. This is measured by the ultrasound machine and converted into an ultrasound image. When a material contains moving parts relative to the source, such as blood flowing past the probe then a frequency shift occurs known as the Doppler principle. This means the reflected waves are at a higher frequency if the object is moving toward the probe and lower if it is moving away. The degree of shift is proportional to the speed of the object and can be detected by the ultrasound probe. The pattern of frequency shifts can be used to derive red cell velocity and then be plotted as a graph demonstrating velocity versus time (Figure 4). In early systole the velocity of the red blood cells is higher causing a higher frequency shift than in late systole and diastole.

The Doppler equation is used to calculate the velocity of red blood cells in the aorta. It requires knowledge of the transmitted (Ft) and the received (Fd) frequencies, the speed of sound in tissue and plasma (C). A correction factor (Cosθ) is used to compensate for the angle of the probe (accurate up to 20°) relative to the axis of the blood flow. Only the reflected frequency shift should be used in calculating the velocity. This means that a factor needs to be included in the equation to halve the velocity to account for the fact that both the transmitted and reflected frequency shifts are picked up.

$$\mathbf{Fd} = \frac{\mathbf{2\ Ft\ V\ Cos\theta}}{\mathbf{C}}$$

To convert the velocity into flow the cross-sectional area of the aorta needs to be included. Most Doppler output monitors use nomograms to estimate this based on height, age, gender and weight. M mode ultrasound can also be used.

Measures of cardiac output: the oesophageal Doppler trace (Figure 4) can be used to infer useful parameters in assessment of cardiac output.

The integral of the velocity/ejection time curve gives us the stroke distance, multiplying this by the estimated cross-sectional aortic area gives us the stroke volume. Multiplying the stroke volume by the heart rate will of course give us the cardiac output.

The mean upslope of the velocity–time graph represents the mean acceleration of red blood cells as a marker of contractility which is independent of vascular tone (unlike velocity).

One of the most useful measures inferred by the velocity time graph is the flow time corrected (FTc) as this is a marker of preload and can be used to guide fluid therapy. The time taken for an entire ejection peak is normalized to a heart rate of 60 bpm. If this is reduced then it suggests a reduced preload and fluid boluses may improve cardiac output. The shape of the trace helps to deduce pathology and cause of reduced cardiac output (Figure 4).

Method of use: select correct probe for duration required (usually in hours or days) and lubricate. Insert orally or nasally to 35–40 cm from teeth and manipulate until Doppler 'whooshing' sound heard with trace on monitor. Adjust gain and cycle length for optimum imaging for interpretation.

Limitations

- The position of the probe is crucial – operator dependent and can move during case/use and lead to change in values.
- Estimation of aortic cross-sectional area has a margin for error.
- Turbulent flow invalidates the equation used to estimate flow from velocity and cross-sectional area.
- Difficult in awake patients.

Benefits

- Well utilized and studied – good comparison to pulmonary artery catheter in terms of trend, less convincing in absolute values.[4]

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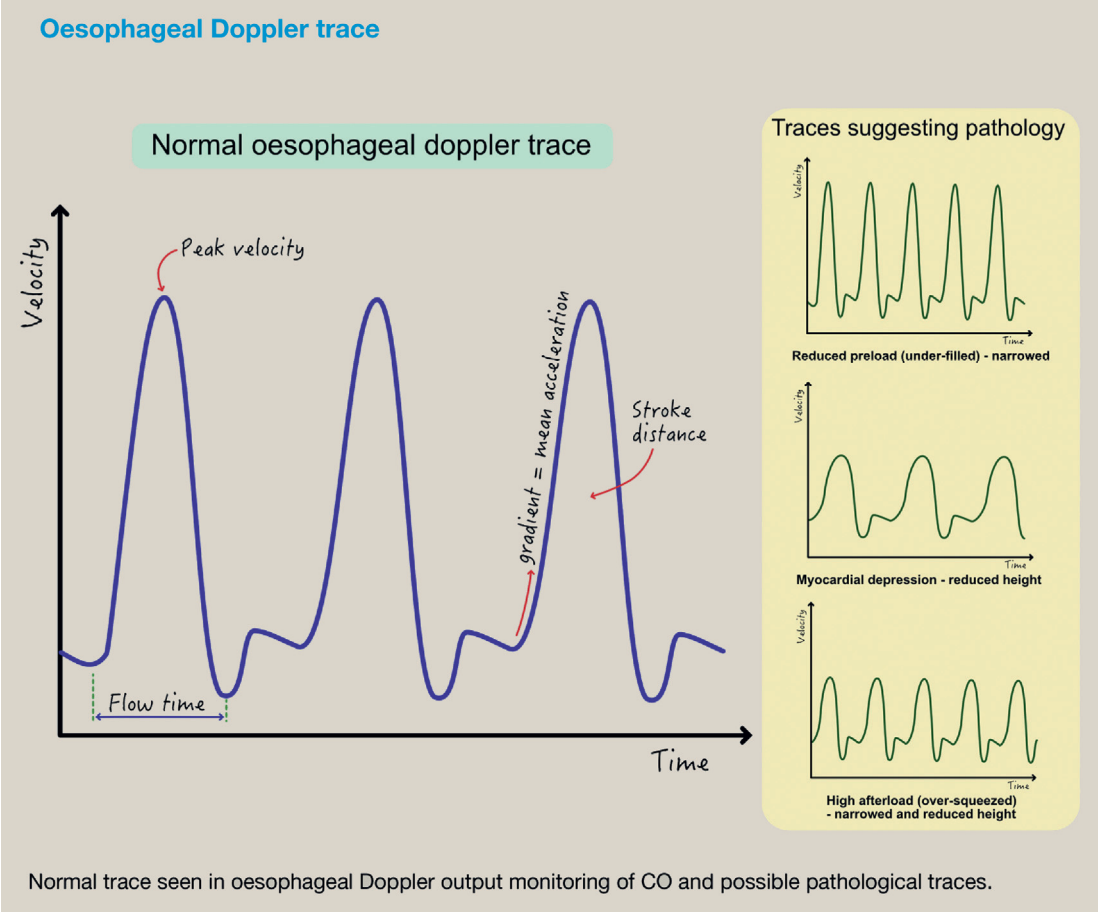
Oesophageal Doppler trace
Normal oesophageal doppler trace
Velocity
Peak velocity
gradient = mean acceleration
Stroke distance
Flow time
Time
Traces suggesting pathology
Reduced preload (under-filled) - narrowed
Myocardial depression - reduced height
High afterload (over-squeezed) - narrowed and reduced height
Normal trace seen in oesophageal Doppler output monitoring of CO and possible pathological traces.

Figure 4

- Less invasive than methods requiring insertion of invasive lines.

Complications

- Oesophageal trauma or stricture formation.
- Probe displacement.

Bioimpedance

Ohm's law and impedance: electrical current can be conducted by charged particles (ions). Ohm's law describes the relationship between potential difference, current (flow of ions) and resistance (to the flow).

Potential difference (V) = Current (I) × Resistance (R)

Therefore, this can be rearranged so that current is equal to potential difference divided by resistance.

I = V/R

Resistance to electrical current is termed impedance when the current is alternating. The term bioimpedance describes the use of this principle to determine impedance by applying Ohm's law and measuring the potential difference when a current is passed through organic tissue. Impedance uses the symbol Z and if current remains constant then changes in resistance and voltage are proportionate.

Different materials including body tissues have different impedance. The bioimpedance model separates high impedance tissues such as air (1274 Ω/cm) and low impedance tissues such as blood (150 Ω/cm). In thoracic bioimpedance (the most commonly used), the thorax is represented as a cylinder with an inner small cylinder representing low impedance blood and a larger cylinder of the higher impedance body tissues. It was later modified to a more conical shape which is more representative of the thoracic cavity.

A high frequency, low amplitude current is passed from outer electrodes at the neck through the thorax to level with the diaphragm while inner electrodes measure the changes. The left and right are averaged to give a mean value. The principle is that the current will find a path of least resistance through the blood filled spaces (represented by the inner low impedance cylinder). This cylinder expands during systole as the aorta distends. This increase in volume leads to a drop in impedance and increase in electrical conductivity. Inner transducing electrodes measure both the baseline impedance (which is inversely proportional to the volume of fluid in the thorax) and the change in impedance. These are timed to the ECG waveform which enables various parameters to be deduced (Figure 5). The measured impedance can be plotted alongside the ECG to find the change in impedance and this change in impedance then plotted. You would expect to see the largest change in impedance after ventricular depolarization representing ventricular ejection and expansion of the

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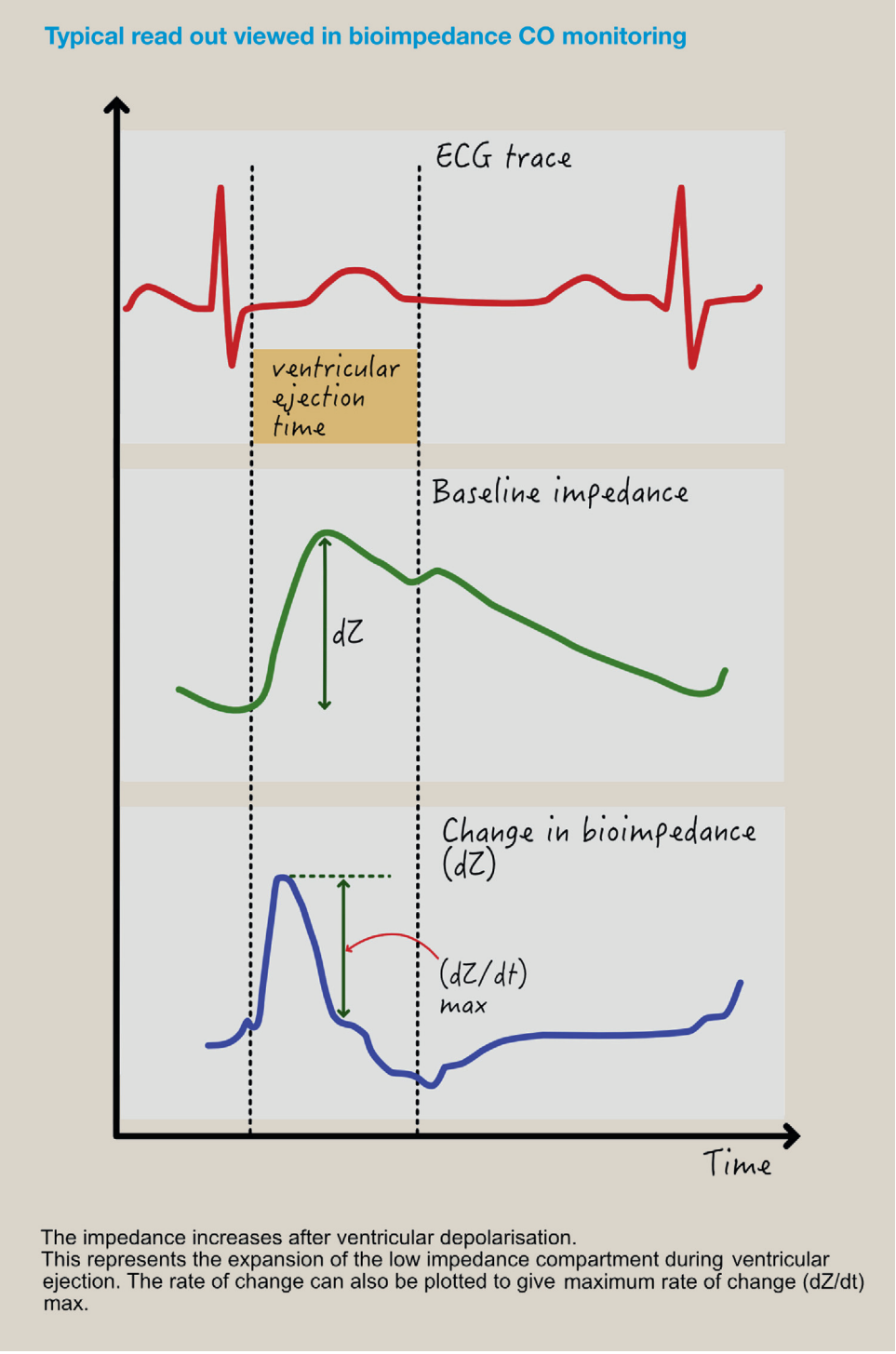
Typical read out viewed in bioimpedance CO monitoring
ECG trace
ventricular ejection time
Baseline impedance
dZ
Change in bioimpedance (dZ)
(dZ/dt) max
Time
The impedance increases after ventricular depolarisation.
This represents the expansion of the low impedance compartment during ventricular ejection. The rate of change can also be plotted to give maximum rate of change (dZ/dt) max.

Figure 5

aorta. The maximum descending slope is termed (dZ/dt)max and is used in the calculation of the ejection phase contractility index (EPCI) by multiplying by the total fluid conductivity.

This index is then used in estimation of the stroke volume. This requires the R–R interval on the ECG to determine the ventricular ejection time (VET) and the volume of electrically participating tissue (or thoracic volume) (VEPT) which is predicted from patient height, weight and gender.

$$\mathbf{SV} = \mathbf{VEPT} \times \mathbf{VET} \times \mathbf{EPCI}$$

This SV can then be used to calculate CO.

Bioreactance

Bioimpedance relies only on measured changes in current amplitude. Bioreactance is an improved technology which analyses relative phase shifts of oscillating currents and the resulting

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voltage signal. This is impacted less by movement artefact, patient body variance and electrode positioning and has had better results in comparative studies.[4]

Method of use: eight electrodes are placed — two either side of the neck/thoracic inlet and two either side of the diaphragm (proximal inner transducing and distal outer current electrodes). Between these electrodes a high frequency, low amplitude current is then passed while the potential difference is measured.

Limitations

- Dependent on placement of electrodes.
- Prone to interference and artefact (e.g. movement, electric diathermy).
- Abnormal fluid in thoracic cavity (e.g. pleural effusion) gives erroneous results.
- Assumptions made in the calculations (no change in vessel capacitance with respiration, all changes in bioimpedance due to aortic blood flow) — margin for error.
- Variable correlation when compared with gold standard — improved with bioreactance technologies.[4]

Benefits

- Non-invasive.

Complications

- Nil significant reported.

Echocardiography

This can be considered a non-invasive or semi-invasive method for assessing both the anatomy and function of the heart.

Method of use: the common methods employed in clinical practice involve the use of an ultrasound probe placed either on the chest wall for a transthoracic echo (TTE) or within the oesophagus for a transoesophageal echo (TOE). Both these forms of assessment give you an image of the heart itself where measurements of a number of structures can be made including the cross-sectional area of the ascending aorta, the left ventricular outflow tract (LVOT) and even the LV volume itself (Figure 6). The velocity time interval (VTi) or pulse wave Doppler can also be observed by using an apical five or three chamber view of the heart. In essence once the area of the LVOT is calculated the pulse wave Doppler/VTi gives a measure of the speed with which the blood is travelling through the LVOT. This generates a velocity time graph, which by using the area under the curve tells you what distance the column of blood has travelled. The volume of this ejected blood (or SV) can be calculated by multiplying the area of the LVOT by the distance the blood travelled. This measured SV can then inform you of the CO.

Benefits

- This method is rapid, effective and relatively non-invasive (certainly with the use of TTE) to determine the CO of patient.
- There is debate as to whether the echocardiography measures up to the 'gold standard' of thermodilution, but a recent meta-analysis has suggested there is no significant difference between these two forms of measurement[5] and that the techniques can be interchangeable in certain circumstances.

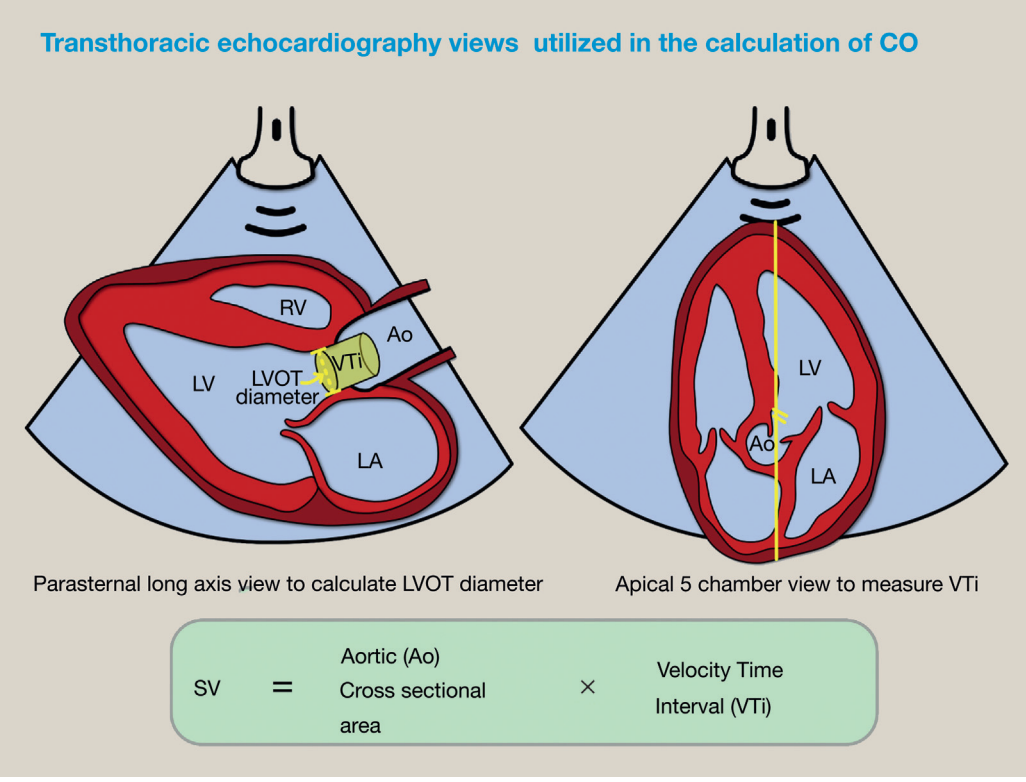

Figure 6

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Limitations

- Use of this tool is limited in certain patient groups, for example patients with a high body mass index are likely to not likely to generate good useable images and can be very user-dependent.
- Requires a significant degree of training and scan time to ensure a user is deemed competent in the use of echo which can limit its availability and speed of assessment.
- Only gives a 'snap shot' window of the patient's cardiac output at that particular time and will need to be reassessed should there be a change or intervention in the haemodynamic state of the patient.
- TOE requires sedation or anaesthetic to allow tolerance of the probe and therefore limits its usability. It is most commonly used intraoperatively for cardiac surgery or on a cardiac ICU.

Complications

- TTE demonstrates relatively few complications beyond reactions to the ultrasound gel.
- TOE is far more invasive and will not only incur the risks of sedation etc but can lead to a risk of damage extending from the teeth, lips and oral cavity to the oesophagus and stomach.
- Serious risks include arrhythmias, bleeding and oesophageal perforation or rupture that would likely require surgical intervention to correct. ◆

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